Analysis of Changes in Reactivity of Rabbit Arteries and Veins Two Weeks after Induction of Hypertension by Coarctation of the Abdominal Aorta

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ABSTRACT

Vessel dimensions and characteristic responses to norepinephrine were measured in various arteries and veins of the rabbit made hypertensive by partial constriction of the upper abdominal aorta. The ear, radial, and basilar arteries taken from the circulation proximal to the ligature (the hypertensive arteries) were thickened in proportion to the rise in arterial blood pressure. The water, sodium, and potassium contents of these and all other vessels were not significantly changed in the hypertensive rabbits. The maximum response to norepinephrine in the ear artery, a representative vessel from the hypertensive part of the rabbit, was increased, whereas the sensitivity of this vessel to norepinephrine expressed as the ED$_{50}$ did not alter with changes in the arterial blood pressure. In contrast, the thickness and the maximum response to norepinephrine of the saphenous artery, representative of vessels distal to the ligature (normotensive vessels) and of the saphenous and cephalic veins were unaltered. The sensitivity as indicated by the norepinephrine ED$_{50}$ of the veins, but not of the saphenous artery, increased with a rise in carotid artery blood pressure. These results suggest that the increased responsiveness to norepinephrine of arteries proximal to the ligature is due to changes in muscle mass and that the increased responsiveness of the veins is due to increased sensitivity to norepinephrine.

The analysis of changes in responsiveness of blood vessels in hypertensive animal models has been the object of many investigations (1-14). A critical survey of these studies led us to define five experimental objectives, which served as the incentive for the present series of experiments. We wanted (1) to accurately determine the effect of hypertension on the responsiveness of vessels to norepinephrine in vitro using vessel rings; (2) to study a variety of vessels, including elastic and muscular arteries and veins, and (3) to separate local consequences of an increased intravascular pressure on the blood vessel wall from any generalized effects associated with the hypertensive state. We felt that (4) small early changes in the vessel wall should be observed if possible, since they are particularly relevant to the genesis of hypertension and that (5) control animal selection should minimize individual variation so that the small changes consequent to the hypertensive state could be recognized.

The experimental model of hypertension which we chose to study was the rabbit with its abdominal aorta partially constricted between the celiac and anterior mesenteric arteries (15-17). In this preparation, after a few days the pressure in the arterial circulation proximal to the ligature becomes raised and that distal to the ligature returns to within the normal range. Although not all of our objectives were realized, many were satisfied by this approach. Existing techniques allowed measurements of vessel responsiveness to norepinephrine in both arteries and veins (18, 19). A comparison of the changes in vessels taken from the proximal and distal parts of the arterial tree allowed changes consequent to an increase in arterial blood pressure to be separated from generalized changes. A comparison of vessels taken distal to the ligature in hypertensive rabbits with those taken from sham-operated rabbits accorded a method of defining generalized circulatory effects. Vessels were studied 2 weeks after constriction of the aorta when the arterial blood pressures in this animal model had been steady for a number of days and the active proliferation of smooth mus-
cle in the vessel walls, although progressing, had reached a peak (17).

In this paper, changes in the response of arteries and veins to norepinephrine are described and partially analyzed. We found that the responsiveness to norepinephrine of both the arteries proximal to the ligature and the veins was increased. The increased responsiveness of the arteries was associated mainly with an increase in the mass of vascular muscle in the vessel walls; in the veins, the alpha receptors became hypersensitive to l-norepinephrine. Preliminary results of the studies on the veins have been previously published (20).

Methods

INDUCTION OF HYPERTENSION

Albino rabbits of either sex weighing 2.25-2.5 kg were starved for 24 hours and then anesthetized with sodium pentobarbital (45-65 mg/kg, ip). Under sterile conditions, polyethylene catheters were inserted into the right femoral artery and into either the right carotid or brachial artery to record arterial blood pressure, utilizing a Statham transducer (P23AA) and a Grass polygraph.

The abdomen was opened at the midline, and a thick silk thread was passed around the abdominal aorta between the celiac and anterior mesenteric arteries. The ligature was tightened until the femoral artery blood pressure several minutes after constriction was approximately half the carotid or brachial artery blood pressure. The abdominal wall was closed in layers without drainage.

The arterial catheters were removed, the catheterized vessels were tied off, and the skin was closed. Sham-operated rabbits were treated in an identical manner except that the abdominal ligature was not tightened before it was tied. Rabbits were fed a commercial pellet diet and given water as required.

Two weeks later, the rabbits were reanesthetized, and catheters were inserted into the left femoral artery and either the left carotid or brachial artery; blood pressures were recorded. The rabbit was then bled through the carotid or brachial artery catheter, and various tissues were rapidly removed and subjected to study. A total of 17 rabbits, including 6 sham-operated animals, was studied. The vessels used included the thoracic aorta and the main pulmonary and basilar arteries, the ear artery proximal to the origin of the arteriovenous anastomoses, the proximal parts of the saphenous and radial arteries, and segments of the cephalic and short saphenous veins distal to the elbow and the knee, respectively.

VESSEL WALL CIRCUMFERENCE AND THICKNESS

Two-mm segments of the ear, radial, basilar, and saphenous arteries and of the cephalic and short saphenous veins were placed in cold Krebs-bicarbonate solution containing phenoxycbenzamine (10⁻³M) and NaN₃ (10⁻³M). (The Krebs-bicarbonate solution was equilibrated with 95% O₂-5% CO₂. The millimolar composition of the solution was: Na⁺ 144.2, K⁺ 4.9, Ca²⁺ 1.3, Mg²⁺ 1.2, Cl⁻ 126.7, HCO₃⁻ 25.0, SO₄²⁻ 1.19, glucose 11.1, and calcium disodium ethylenediaminetetraacetate (EDTA) 0.024. The pH of the solution was 7.4.) These agents were used to remove any catecholamine-induced or myogenic tone that might be present, although no attempt was made to measure the tone. This procedure was necessary to permit a meaningful comparison of vessel thickness. One hour later, the flaccid vessels were cut longitudinally, laid flat, the intimal surface downward, on a glass slide inscribed with a calibrated scale. The inner circumference was measured against the scale marked by DMI (10⁻³M), tert butyl alcohol, and dibutyryl cyclic AMP. The outer circumference was measured with fine calipers. Steady-state contractile responses to /-norepinephrine (0.12 µg, 95% O₂-5% CO₂) were measured. (4) After the vessels had been soaked in desmethylimipramine (DMI, 10⁻⁵M) for 30 minutes, the cumulative addition of norepinephrine was repeated and a final concentration of norepinephrine of 0.12 mM was added to obtain a maximum response (the norepinephrine maximum response is unaltered by DMI [10⁻⁷M], Bevan, unpublished data). (5) Finally, tissues were wiped in a standard manner on a glass tile, and the slope of the dose-response curve was obtained in the absence and the presence of DMI; from vessel dimensions and weight, cross-sectional area was calculated.

ORGAN WEIGHT AND HISTOLOGY

In addition to a general postmortem examination, the heart and kidneys were weighed. Histological examination of the kidneys was carried out.

VESSEL WATER, SODIUM, AND POTASSIUM CONTENT

Vessel rings were opened by a longitudinal cut; the tissues were wiped in a standard manner on a glass tile and transferred to oven-dried hard glass tubes for weighing. The loss of weight after overnight heating at 105°C provided the water content. The desiccated tissue was digested in 0.2 ml of concentrated nitric acid and diluted; then the sodium and potassium contents were estimated on a flame photometer against external standards.

IN VITRO TESTING OF REACTIVITY

Rings of vessels 2 mm long were set up for isometric recording of the contraction in vitro (18). A resting tension previously determined to be optimal for the particular vessel in the normotensive rabbit was used. For the ear and saphenous arteries this tension was 0.50 g, and for the cephalic and saphenous veins it was 0.25 g. After equilibration for 30 minutes in Krebs-bicarbonate solution, the following schedule of experimental measurements was carried out. (1) Vessel length and circumference were measured with fine calipers. (2) Steady-state contractile responses to l-norepinephrine (0.12 µM; this dose is close to the norepinephrine ED₅₀ of the vessels studied) were determined. (3) Responses to 3-4 cumulative doses of l-norepinephrine to a level not exceeding the ED₅₀ were measured. (4) After the vessels had been soaked in desmethylimipramine (DMI, 10⁻⁵M) for 30 minutes, the cumulative addition of norepinephrine was repeated and a final concentration of norepinephrine of 0.12 mM was added to obtain a maximum response (the norepinephrine maximum response is unaltered by DMI [10⁻⁷M], Bevan, unpublished data). (5) Finally, tissues were opened by a longitudinal cut, wiped in a standard fashion on a glass tile, and weighed on a Cahn electrobalance.

From these data, the probit regression of percent maximum contraction on log dose of norepinephrine, the ED₅₀, and the slope of the dose-response curve was obtained in the absence and the presence of DMI; from vessel dimensions and weight, cross-sectional area was calculated.

Circulation Research, Vol. 37, August 1975
**HYPERTENSIVE ARTERIES AND VEINS**

**DATA ANALYSIS**

The relationships among all of the parameters were examined by plot-scatter diagrams of parameters paired from each individual rabbit. Pearson's product-moment correlation coefficient was computed whenever a linear trend was indicated. The $P$ value for testing the statistical significance of the correlation coefficient was determined from Table A-30a of Dixon and Massey (21).

**Results**

Among the 17 rabbits included in this study, 6 sham-operated and 11 aorta-ligated, the highest carotid artery blood pressure at the end of the 2-week period was 160 mm Hg. The greatest carotid-femoral artery pressure difference was 75 mm Hg: the mean carotid and femoral artery pressures of the operated rabbits, 127 ± 4 and 101 ± 4 (SEM) mm Hg, respectively, were significantly different ($P < 0.01$). This mean femoral arterial pressure was slightly but not significantly greater than the preoperative mean femoral artery pressure of all of the rabbits, 89 ± 3 mm Hg.

Since the mean increase in carotid artery blood pressure with surgery was variable and small, the results from all of the rabbits in the series, both operated (aorta-ligated) and sham-operated, were grouped together, and usually the measured parameter was plotted as a function of either carotid artery pressure, femoral artery pressure or the carotid-femoral artery pressure difference.

During the course of these experiments, it became obvious that isolated segments of arteries and veins from hypertensive rabbits developed a greater contractile response than did those from sham-operated rabbits. An analysis of the changes in arteries and veins will be presented separately later.

With the exception of one rabbit, which was eliminated from the series, no gross pathology was noted. Heart weights increased with the rise in arterial blood pressure. Kidney weights were independent of arterial blood pressure.

**CHANGES IN ARTERIES PROXIMAL TO THE AORTIC LIGATION**

There was a definite trend for the wall thickness of the basilar and ear arteries to increase when the carotid artery blood pressure increased. The correlation coefficients (95% confidence limits) were 0.76 (0.40 to 0.9) and 0.65 (0.2 to 0.87), respectively. In the radial artery, the same trend was present, but it was not significant (Table 1). The measurement of wall thickness was technically most satisfactory and accurate in the basilar artery, since both surfaces of this vessel are biologically defined by the intima and the arachnoid. The mean of the coefficients of variation of wall thickness obtained from measurements of each of the 17 basilar arteries was 0.09. In other vessels, the outer adventitial surface was demarcated in part by dissection. Presumably this fact contributed to the variation in the measured thickness. In the ear artery, the mean of the coefficients of variation was 0.14. In Figure 1, the relationship between wall thickness and carotid arterial blood pressure is shown for the basilar artery. Except for three

**TABLE 1**

*Selected Correlation Coefficients between Various Blood Vessel Parameters and Carotid Arterial Blood Pressure of Rabbits with Partial Constriction of the Upper Abdominal Aorta and of Their Sham-Operated Controls*

<table>
<thead>
<tr>
<th>Vessel</th>
<th>Ear artery</th>
<th>Basilar artery</th>
<th>Radial artery</th>
<th>Thoracic aorta</th>
<th>Saphenous artery</th>
<th>Cephalic vein</th>
<th>Saphenous vein</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$r$</td>
<td>$P$</td>
<td>$r$</td>
<td>$P$</td>
<td>$r$</td>
<td>$P$</td>
<td>$r$</td>
</tr>
<tr>
<td>Wall thickness</td>
<td>0.653</td>
<td>$&lt;0.01$</td>
<td>0.756</td>
<td>$&lt;0.01$</td>
<td>0.303</td>
<td>$&gt;0.1$</td>
<td>0.110</td>
</tr>
<tr>
<td>Internal circumference</td>
<td>0.28</td>
<td>$&gt;0.1$</td>
<td>0.025</td>
<td>$&gt;0.1$</td>
<td>0.149</td>
<td>$&gt;0.1$</td>
<td>0.309</td>
</tr>
<tr>
<td>Maximum</td>
<td>0.680</td>
<td>$&lt;0.01$</td>
<td>-0.416</td>
<td>$&gt;0.1$</td>
<td>0.424</td>
<td>$&gt;0.1$</td>
<td>0.475*</td>
</tr>
<tr>
<td>Norepinephrine response</td>
<td>-0.239</td>
<td>$&gt;0.1$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.146</td>
</tr>
<tr>
<td>Percent H$_2$O</td>
<td>0.201</td>
<td>$&gt;0.1$</td>
<td>0.241</td>
<td>$&gt;0.1$</td>
<td>0.487</td>
<td>$&gt;0.1$</td>
<td>-0.472*</td>
</tr>
<tr>
<td>K</td>
<td>0.154</td>
<td>$&gt;0.1$</td>
<td>0.165</td>
<td>$&gt;0.1$</td>
<td>0.244</td>
<td>$&gt;0.1$</td>
<td>0.537</td>
</tr>
<tr>
<td>Na</td>
<td>-0.246</td>
<td>$&gt;0.1$</td>
<td>-0.014</td>
<td>$&gt;0.1$</td>
<td>-0.091</td>
<td>$&gt;0.1$</td>
<td>0.085</td>
</tr>
</tbody>
</table>

$r$ = correlation coefficient and $P$ = probability of slope of regression different from zero. The number of rabbits studied varied between 15 and 17.

*Correlation with femoral arterial blood pressure.

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rabbits, the arterial wall thickness of the basilar artery could be predicted quite accurately from the carotid arterial blood pressure. Over the arterial blood pressure range studied (100 to 160 mm Hg), the mean arterial wall thickness more than doubled.

**Internal Circumference.**—In all of the arteries (basilar, ear, and radial) studied, the correlation coefficients between the internal circumference (and hence the diameter) and the arterial blood pressure were small.

**Maximum Response to l-Norepinephrine in the Ear Artery.**—The central ear artery, a muscular artery which has been the object of considerable investigation (22), was selected to represent arteries proximal to the ligature. The positive correlation coefficient between the maximum response to norepinephrine and the mean carotid arterial blood pressure was large, 0.68 (0.28 to 0.87) (Fig. 2, top). In this vessel (within the 100–160 mm Hg range), the mean increases in the maximum response to norepinephrine and the wall thickness were on the order of 66% and 85%, respectively. Presumably, an increased amount of muscle in the artery wall was responsible for the increased responsiveness. This possibility was supported by the plot of maximum contractile response divided by vessel cross-sectional area vs. carotid arterial blood pressure (Fig. 2, bottom). When maximum developed tension (contractile response divided by vessel cross-sectional area) was plotted against carotid arterial blood pressure, although the correlation coefficient was only $-0.42 (-0.75$ to $1.0)$ and was not significantly different from zero statistically, the scatter diagram did suggest that an increase in carotid artery blood pressure was, in fact, associated with a small decrease in the ability of the artery wall to develop contractile tension.

**Characteristics of Norepinephrine Responses.**—For most rabbits, the norepinephrine $ED_{50}$ of the ear artery determined in either the presence or the absence of DMI did not change with arterial blood pressure (Table 1, Fig. 3). Since the $ED_{50}$...
HYPERTENSIVE ARTERIES AND VEINS

FIGURE 3
Relationship between the mean carotid artery blood pressure and the median effective dose of norepinephrine (NE ED₅₀) for the contractile response of ring segments of the ear artery taken from rabbits with partial constriction of the upper abdominal aorta and from their sham-operated controls.

was derived from a fitted regression line and might not accurately reflect changes in response to just-suprathreshold doses of norepinephrine, the relationship between the developed force of the ear artery to a low, suprathreshold dose of norepinephrine (8.0 × 10⁻⁹ M) expressed as a percent of the maximum force and the carotid arterial blood pressure was examined. There was a definite trend for the contractile response to norepinephrine in the presence or the absence of DMI to decrease with an increase in the arterial blood pressure. The correlation coefficients for the contractile response of the ear arteries with and without DMI were -0.729 (-0.9 to -0.40) and -0.60 (-0.78 to -0.1), respectively. The corresponding P estimates were < 0.01 and < 0.05. These findings indicate that as the carotid artery blood pressure rises, the response to a just-suprathreshold concentration of norepinephrine decreases.

CHANGES IN ARTERIES DISTAL TO THE AORTIC LIGATURE

Characteristics of the response to norepinephrine, the ED₅₀, and the maximum contractile response did not show a significant correlation with either carotid artery pressure, femoral artery pressure, or the carotid-femoral artery pressure difference (Table 1).

Characteristics of the Norepinephrine Response.—There was a highly significant negative correlation between the norepinephrine ED₅₀ and the carotid artery blood pressure in both the cephalic and saphenous veins (Fig. 4, Table 1). In the saphenous vein, for example, based on the regression line, veins taken from rabbits with a carotid artery pressure of 160 mm Hg would, on the average, be expected to be six times more sensitive to norepinephrine than those taken from normotensive rabbits.

WATER AND ION CONTENT OF BLOOD VESSELS

In none of the vessels studied was there a correlation between water, sodium, and potassium content and carotid or femoral artery blood pressure (Table 1). There was no significant difference between the content of the ions and water in pooled ear and saphenous arteries. The potassium content of the pooled saphenous veins was smaller than that in other vessels studied with the exception of the thoracic aorta (Table 2).

Discussion

Partial constriction of the abdominal aorta above the origin of the renal arteries, after a latency of several days, results in elevation of arterial blood pressure in the circulation proximal to the constriction and normotensive conditions below the constriction in arteries distal to the aortic ligature.

CHANGES IN VEINS

Segments of the cephalic and saphenous veins, from the upper and lower limbs, respectively, were studied. Vein wall thickness and internal diameter did not significantly change in hypertensive rabbits, nor was there a significant correlation between the maximum response to norepinephrine and carotid artery pressure, femoral artery pressure, or the carotid-femoral artery pressure difference (Table 1).

FIGURE 4
Relationship between the mean carotid artery blood pressure and the median effective dose of norepinephrine (NE ED₅₀) for the contractile response of ring segments of the saphenous vein taken from rabbits with partial constriction of the upper abdominal aorta and from their sham-operated controls.
striction. Although this model is not associated with an elevation in plasma renin levels (16), there is some evidence that the kidneys are essential for the development of the hypertension (for references with an elevation in plasma renin levels (16), there is the development of the hypertension, see Nolla-Panades [3]). Rabbits were studied 2 weeks after constriction, since by that time arterial blood pressure had reached a plateau (17). Arterial blood pressure was measured during sodium pentobarbital anesthesia. Although the anesthetic might alter the absolute level of anesthesia was utilized in all of the rabbits. Furthermore, essentially similar results were obtained when the measured parameters were plotted against the carotid-femoral artery blood pressure difference; the anesthetic presumably influenced both pressures in a similar manner. Wall thickness changes were measured under conditions for which intrinsic tone had presumably been reduced to a minimum. A raised arterial blood pressure is almost invariably associated with changes sufficient to account for increases in wall thickness of 30–50% would be readily apparent. The most likely conclusion is that cellular changes must account for the remarkable increase in vessel wall thickness. The previously cited studies show that proliferation is mostly confined to the media, leading to the conclusion that vessel wall thickening must be due to hyperplasia or hypertrophy of the vascular smooth muscle.

This conclusion was supported by the observation that the maximum response to norepinephrine increased significantly with a rise in arterial blood pressure in the absence of changes in the ED<sub>50</sub>. It is of interest that the maximum response to norepinephrine divided by wall thickness (at the best, a poor measurement of muscle cell content) showed a trend toward a negative regression. In this context, Shibata et al. (24) have found that the maximum response of the aorta of spontaneously hypertensive rats to norepinephrine is reduced over controls. Similar findings have been reported by Clineschmidt et al. (6), Levy (11), and Spector et al. (25). The rat aorta does not increase in thickness with a rise in arterial blood pressure. However, such a decrease in maximum response is not seen with all agonists (Levy [11]). A fall in maximum response may be associated with the disruption of contractile elements (26); alternatively, since the same resting tension was used for all specimens of a particular vessel irrespective of the arterial blood pressure of the donor animal, the tension could have been suboptimal for thicker vessels. Such an effect could account for the trend toward a fall-off in the maximum response in vessels from hypertensive animals. In small blood vessels there is some evidence for an increased maximum response to various agonists in hypertension (14, 27). It would seem that the final measured maximum contraction in a blood vessel must be the net result of a number of factors, including changes in the num-

### Table 2

<table>
<thead>
<tr>
<th>Vessel</th>
<th>No. of rabbits*</th>
<th>H₂O (%)</th>
<th>Potassium (μg/100 g wet wt)</th>
<th>Sodium (μg/100 g wet wt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ear artery</td>
<td>13 (9 + 4)</td>
<td>73.2 ± 0.9</td>
<td>171 ± 14.9</td>
<td>371.9 ± 17.8</td>
</tr>
<tr>
<td>Radial artery</td>
<td>12 (8 + 4)</td>
<td>72.0 ± 2.6</td>
<td>243 ± 19.2</td>
<td>352.2 ± 40.5</td>
</tr>
<tr>
<td>Thoracic aorta</td>
<td>12 (8 + 4)</td>
<td>75.0 ± 1.4</td>
<td>65.8 ± 12.2</td>
<td>273.5 ± 28.6</td>
</tr>
<tr>
<td>Saphenous artery</td>
<td>10 (7 + 3)</td>
<td>73.8 ± 0.9</td>
<td>197 ± 13.3</td>
<td>388.4 ± 55.8</td>
</tr>
<tr>
<td>Saphenous vein</td>
<td>4 (3 + 1)</td>
<td>76.0 ± 4.1</td>
<td>60.5 ± 11.6</td>
<td>457.2 ± 28.6</td>
</tr>
<tr>
<td>Cephalic vein</td>
<td>5 (3 + 2)</td>
<td>75.6 ± 2.0</td>
<td>120.4 ± 12.1</td>
<td>428.8 ± 30.1</td>
</tr>
</tbody>
</table>

All values are means ± SE. There was no significant correlation between values from individual rabbits and carotid arterial blood pressure (see Table 1).

* Total number of rabbits (ligated + sham-operated).
ber or size of the muscle cells, the contractility of the cellular elements, and the biochemical and coupling mechanisms (24, 28, 29).

The internal circumference of vessels examined in this study was unaltered. In muscular arteries in this model, intimal proliferation does not occur (17), although such changes do occur in other blood vessels, particularly the arterioles. It would seem reasonable that the internal circumference in the relaxed muscular artery after short-term hypertension would be determined by the internal elastic lamina together with other elements. Folkow et al. (9) and Sivertsson (23) have proposed, on the basis of pressure-flow studies, that the internal circumference of resistance vessels is reduced in spontaneously hypertensive rats. Since in these vessels subintimal proliferation is frequently observed (17), although such changes do occur in other blood vessels in hypertension has been observed by many investigators in a number of experimental models (13-14, 24, 29). In general, the changes found by these workers were small. In the present experiments, the increased sensitivity to norepinephrine was highly significant and occurred in vessels (veins) in which the intraluminal pressure was low compared with the arterial blood pressure and was unchanged in this model of hypertension (Bevan et al., unpublished results).

As there is some suggestion that there are generalized influences of possible renal origin in this model responsible for the hypertension, it might be reasonable to assume that the increased venous sensitivity is related to these changes. If the increased sensitivity of the veins were due to some generalized effect, then there is no reason why similar changes should not take place in the arteries. This was not the case. The norepinephrine ED50 of the ear artery was independent of any measured arterial blood pressure. The ED50 of the saphenous artery could not be significantly correlated with carotid or femoral artery blood pressure. It could be argued that the increased pressure in the proximal arteries was responsible for a generalized decrease in reflex sympathetic drive. Although pressoreceptor resetting has been documented in hypertensive states, its place or role in this particular model 2 weeks after ligation is unknown. Such a functional neuronal inactivity might lead to increased receptor sensitivity and would be somewhat equivalent to decentralization hypersensitivity (31). It is not known, however, whether a change of this degree could occur as a consequence of decreased sympathetic activity and why this mechanism would not affect the arteries as well as the veins. The change in venous sensitivity might be associated with alterations of coupling or biochemical mechanisms (13, 14, 24, 28).

Our data on water and ion content do not support any theory based on differences in total ion content in this model. They are consistent with the observations of others (32, 33) in the spontaneously hypertensive rat and of Jones (34), who found alterations in ion kinetics rather than ion content with hypertension in rats. In blood vessels of other animal models of hypertension, there are increases in water, sodium, and potassium content in renal hypertension (35, 36), in deoxycorticosterone hypertension (36), and in adrenal regeneration hypertension (37).

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